AMENDMENT TO THE SPECIFICATION

Please replace the paragraph bridging pages 1 and 2 with the following amended version of that paragraph:

Asthma is a reversible obstructive pulmonary disorder caused by an airway hyper-responsiveness to specific and/ or non-specific stimuli. Asthmatic airway obstruction typically results from bronchospasms. Asthma may be triggered by a variety of causes such as allergic reactions, a secondary response to infections, industrial or occupational exposures, ingestion of certain chemicals or drugs, exercise, and vasculitis. Much of asthma's pathology can be attributed to mast cell degranulation. Mast cells will degranulate in response to various conditions such as, for example, classical IgE-antigen stimulation. It is believed that when the asthmatic, human or animal, inhales an allergenic substance, sensitized IgE antibodies trigger mast cell degranulation in the lung interstitium. The mast cell degranulation releases histamine, bradykinin, and slow-reacting substance of anaphylaxis (SRS-A) which includes the leukotrienes C, D and E, prostaglandins including PGF_2, PGF_{2a}, and PGD_2, and thromboxane A_2. The histamine then attaches to receptor sites in the larger bronchi, causing irritation, inflammation, and edema. The SRS-A attaches to receptor sites in the smaller bronchi, causing edema and attracting prostaglandins, which enhance the effects of histamine in the lungs. Histamine, in combination with prostaglandins, also stimulates excessive mucous secretion, narrowing the bronchial lumen further. When an asthmatic individual inhales, the narrowed bronchial lumen still expands slightly, allowing air to reach the alveoli. However, upon exertion to exhale, the increased thoracic pressure closes the bronchial lumen completely. Therefore, air can enter the lungs, but may not exit during an asthma attack. The ventilation in the alveoli is then inhibited by mucous collecting in the lung bases. In an effort to compensate for lowered alveolar ventilation, blood is shunted to other alveoli. Hypoxia, and in extreme cases, respiratory acidosis may result without medical intervention. In many cases, there are two phases to an allergic asthma attack, an early phase and a late phase which follows 4-6 hours after bronchial stimulation. The early phase includes the immediate inflammatory response including the reactions caused by the release of cellular mediators from mast cells (i.e., histamine). Late phase reactions develop over a period of hours and are characterized histologically by an early influx of polymorphonuclear leukocytes and fibrin deposition, later followed by infiltration of eosinophils. Increased levels of eosinophil-derived inflammatory mediators in plasma and BAL, including eosinophilic cationic protein and major basic protein, have been observed during the late phase reaction. Upregulation of TH2-type cytokines ([[IL4)] IL-4, [[IL5]] IL-5 and [[IL 13]] IL-13) following allergen challenge has also been observed during the late phase. Thus, the cellular inflammatory response, in combination with released pro-inflammatory mediators (e.g., mmp9) and locally produced cytokines in the bronchial mucosa, play a central role in the late phase allergic inflammation and bronchoconstriction. Late phase reactions increase airway reactivity and lead to prolonged asthmatic exacerbations that may last from hours to days to months in some subjects. One of the residual effects of asthma reactions is this hyperresponsiveness of the airways to nonspecific stimuli.

Please replace the paragraph on page 6, lines 8-18, with the following amended version of that paragraph:

A combination therapy may also be used that includes a complement-inhibiting compound in combination with a regimen of known asthma therapy, such as, for example, steroids, anti-IgE antibodies, anti-IL-4 antibodies, anti-IL-5 antibodies, β2 receptor agonists, leukotriene inhibitors, 5 Lipoxygenase inhibitors, b2 adreno receptor agonists, PDE inhibitors, [[IL 5]] IL-5 antagonists, CD23 antagonists, [[IL 13]] IL-13 antagonists, cytokine release inhibitors, histamine H1 receptor antagonists, anti-histamines and histamine release inhibitors. Suitable compounds of each class listed above as well as other asthma treatments are listed in Asthma Therapeutic: New Treatment Options and Emerging Drug Discovery Targets ~~Tasrgerts~~, Barnes, April 2003, LeadDiscovery~~,~~ ~~http://www.leaddiscovery.co.uk/target-discovery/abstracts/dossier-asthma.html~~.

Please replace the paragraph on page 22, lines 5-11, with the following amended version of that paragraph:

The BB5.1 antibody is made according to known methods. (See, Frei, Y., Lambris, J. D., Stockinger, B. Mol. Cell. Probes. 1: 141-149 (1987)). Both the BB5.1 antibody and the isotype match control 135.8 hybridoma antibody were grown as ascites in athymic mice and the antibodies were purified from ascites by protein A affinity chromatography followed by elution with ~~ImmunoPure~~ ImmunoPure® IgG elution buffer (Pierce) and dialysis against PBS buffered saline. (Wang et al. 1996).